Awaking a sleeping epidemic

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Two patients with African sleeping sickness (SS) presented to the neurology unit, Pretoria Academic Hospital, during 2004 and 2005. SS has shown a recent resurgence, with epidemics in the Sudan, Angola and the Democratic Republic of Congo. The number of infected people in Africa is currently estimated at more than 500 000. According to the World Health Organization (WHO), about 20 *Trypanosoma brucei gambiense* and 30 *T. b. rhodesiense* infections are diagnosed yearly outside endemic areas in Africa. Migration, tourism, peacekeeping and military interventions and the re-emergence of SS epidemics might increase these numbers.¹

The electroencephalogram (EEG) is often useful in the diagnosis of coma and delirium, but has not been widely used in the diagnosis of SS. The EEG is proposed as a novel way to follow disease progression, treatment response and treatment-induced encephalopathy.

Case 1

A 27-year-old man presented with a 4-month history of fatigue, loss of appetite, intermittent severe headaches, excessive daytime sleepiness, loss of concentration and insomnia. He had travelled to Malawi 8 months before admission. His temperature was 38.8°C, he had a palpable hepatomegaly and an unremarkable neurological examination although his cognitive response was slow. Diagnosis of African trypanosomiasis was made on a Giemsa-stained blood smear (Fig. 1).

Shortly after admission the patient had a tonic-clonic seizure, with post-ictal confusion. Treatment with suramin was started and repeat blood smears after 48 hours were clear of trypanosomes. The cerebrospinal fluid (CSF) showed no trypanosomes but a total protein level of 1.2 g/l, glucose 2.1 mmol/l, 4 polymorphs and 82 lymphocytes. WHO-recommended treatment with melarsoprol was started.²

Since no trypanosomes were isolated from inoculated mice the diagnosis of West African trypanosomiasis (WAT) was made. Effornithine was unavailable and treatment with melarsoprol continued. The patient recovered well and returned to the UK.

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Five months after discharge he presented to the Hospital for Tropical Diseases in London with fever, sleepiness and an active CSF. Diagnosis of a relapse was made which posed a diagnostic dilemma – recurring *T. b. gambiense*. Treatment with effornithine was given which cleared his condition.

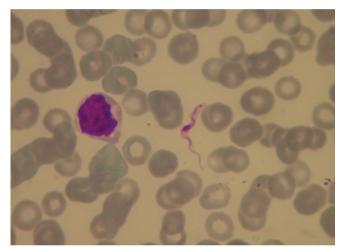


Fig. 1. Giemsa smear (case 1) showing extracellular trypanosomiasis parasite in the peripheral blood.

Case 2

A 53-year-old man presented with a 2-week history of fever, headache and episodic confusion. He was a farmer from Kariba in Zimbabwe where he had been treated for malaria without any clinical improvement. He gave a history of multiple tsetse fly bites but did not have a chancre. A Giemsa-stained blood smear showed *Trypanosoma* spp. On admission his temperature was 39.4°C, but the general examination was unremarkable. He was very sleepy but easily arousable. The diagnosis of East African trypanosomiasis (EAT) was confirmed by isolating *T. b. rhodesiense* from inoculated mice. Treatment with suramin was started and repeated Giemsa-stained blood smears did not show any trypanosomes. The following day he had a fatal cardiac arrhythmia, probably due to myocarditis.

Our first patient had a series of EEG recordings. These indicated a low-voltage mixed-frequency background with episodic, generalised but frontally dominant irregular delta activity (Fig. 2). Follow-up showed a gradual improvement in the frequency of the background. The second patient had an EEG recording shortly after admission showing mild slowing of the background activity with similar episodes of irregular frontally dominant delta activity.